Dear Phil:

I have been meaning to write you for weeks and weeks, but it would be an understatement to say that things have been in a turmoil here. We had some very good news today though, that makes me want to catch up on min all these unfilled tasks— one of my students was in a terribly serious auto accident two weeks ago, but is just now coming out of the woods.

First of all, let me thank you for the paralyzed S. typhi. It certainly is what you say, perfectly stable non-motile, but with a strong agglutination in d-serum. Is it the routine to test for motility of a Salmonella before routine H- agglutination tests? Anyhow I have tried (as you did no doubt also) to transduce motility to it, but so far none of my reagents have been effective. It is impossible to draw any inferences from this until there have been some attempts to transduce other characters; it may be that the strain either does not respond at all to the PLT22 reagents, or that its paralysis depends on two different genetic loci. I was very happy to have this thrain, and will indeed rely on you for any other non-motile variants that may accumulate.

About the Serratia, the baby girl is still exceeting it. However, since she went off a pre-serilized diet, she picked up a normal coliform flora as well and has only a small proportion (c. 1/1000) of the red colonies on nutrient agar plates. The incidence of Serratia-carriers at that level may be higher than one could readily guess. Her diapers now turn only a light pink instead of the initial dark red.

Dr. Bernstein is now in the middle of his study of crosses of Olll, etc. Of a large series of strains tested so far, the only fertile cultures have been an 0551 that Esther had screened from Bill Ewing's collection just after our visit; and an 0-26 from Manchester. The fertile 0-55 strain is your number 589/52. If you have any history that should be recorded with it, we would be glad to have that. It might be fun, if there were other isolations from the same outbreak, to see if they were also fertile. But it would be of more scientific importance to be able to screen a larger number of 0-55's and 0-111's to try to find better material for genetic study. The 0-111 strain 808/50 (#95) had us going for a while, but proved to memutating to streptomyfin-resistance rather frequently, rather than crossing as we thought from the preliminary tests. At any rate, the 0-26 and wall 0-55 strains are being crossed with strain K-12, from which tests they can be said to be of the "F- mating type". Unfortunately, they are somewhat resistant to being converted to the "F+ mating type", and this has been an impediment against the most desirable cross of xx 0-26 x 0-25 (since the K-12 is rather rough.) We are getting somewhat low on the relevant coli sera, but will hang on for a while before making mi further appeal.

There are, however, a couple of favors that I would very much apprepriate. I wrote you once before about the relationship of phage infection to the IV 4,12 — 4,5,12 changes in, e.g., S. abortus—equi as you picked up in our earlier H transductions. There is something peculiar about the 5 reaction here, or perhaps about the 4,12 typhimurium strain that we were using for adsorption. At any rate, just to help sort out our cultures, it would be a tremendous help if we could have a small addl. sample of your "standard 5" serum, which was able to distinguish the two groups of cultures initially. We should need enough for about 40-50 slide agglutination tests. If we can sort these out, we can then make wise choices for further immunizations and absorptions.

My other request is for some Salmonella stocks: 1) S, virginia, which I read as carrying H₁^d similar to typhi, but not cross-reacting with typhi-0 serum [for an experiment with fluorescent antibodies to distinguish H and O reactions on living cells], and 2) whatever, I trust little, you may have that carries Salmonella 35, in the light of Olarte and Varela's identification of Salmonella 35(3. adelaide) ; E. coli OlliB4.

See yours
of 9/24/! I hardly know what to say about your mentioned observations on changes incidental to meremperate changes in H factors. Phage might have something to do with it, but I can't see by what pattern just at the moment. I just don't have a clear enough picture of the present story to beaable to speculate very clearly (which is doubtless why I have overlooked answering the point).

Have you ever gotten your reprints from Iseki? I don't believe I have any that were intended from you, but I am sure he would respond immediately to your own request.

Yours sincereky.

Joshua Lederberg

P.S. We have a lead on the source of that Serratia (which will make it next thing to a lab. infection). Would the serology be able to confirm the identity of the fecal isolate with other strains? Would this be interesting enough to be worth doing? (There seems to be a lab across the street that may be disseminating moderate quantities of Serratia into the atmosphere).